Esophageal Cancer Among Black Men in Washington, D.C. I. Alcohol, Tobacco, and Other Risk Factors [1]

Linda M. Pottern, *M.P.H.*, [2] Linda E. Morris, *M.P.H.*, [2] William J. Blot, *Ph.D.*, [2] Regina G. Ziegler, *Ph.D.*, [2] *and* Joseph F. Fraumeni, Jr., *M.D.* [2,3]

ABSTRACT—A case-control study involving interviews with the next of kin or close friends of 120 black males who recently died of esophageal cancer and 250 similarly aged black males who died of other causes was undertaken to discover reasons for the exceptionally high mortality from this cancer in Washington, D.C. The age-adjusted annual death rate in Washington, D.C., for nonwhite males, 1970–75, was 28.6/100,000, far higher than the national rate of 12.4/100,000 and the rates in other metropolitan areas of the country. The major factor responsible for the excess was alcoholic beverage consumption, with an estimated 81% of the esophageal cancers attributed to its use; high use of alcoholic beverages was also found among the controls. The relative risk (RR) of esophageal cancer associated with use of alcoholic beverages was 6.4 (95% confidence interval=2.5, 16.4). The RR increased with amount of ethanol consumed and was highest among drinkers of hard liquor, although the risk was also elevated among consumers of wine and/or beer only. The RR associated with cigarette smoking was 1.9 (1.0, 3.5) when controls with smoking-related causes of death were excluded but declined to 1.5 (0.7, 3.0) when adjusted for ethanol consumption. Significant differences of approximately twofold were found between low and high levels of *a*) consumption of fresh or frozen meat and fish, fruits and vegetables, and dairy products and eggs and *b*) relative weight (wt/ht^2). The inverse trends with these general measures of nutritional status were not explained by alcoholic beverage consumption or socioeconomic status as measured by educational level.—JNCI 1981; 67:777-783.

Mortality rates for esophageal cancer among black male residents of Washington, D.C., are among the highest in the United States (*1*). To identify the risk factors that may be responsible, we conducted a case-control interview study with the next of kin of black males who had died of esophageal cancer.

MATERIALS AND METHODS

To update esophageal cancer mortality statistics reported for 1950–69 (*1*), we computed mortality rates for 1970–75 by sex, race (white, nonwhite), and age for D.C., other metropolitan areas, and the entire United States, with the use of data from the National Center for Health Statistics and the Bureau of the Census. Deaths for 1972 were not recorded at the national level and are not included in the calculations. Methods of calculation for age-adjusted rates are described in (*1*).

Subjects for the case-control study were identified from a computerized mortality tape from the D.C. Department of Human Resources. All deaths among black male residents attributed to primary esophageal cancer [code No. 150 (*2*)] during the years 1975–77 were selected as cases. Controls were randomly selected from among other causes of death (excluding oral, pharyngeal, and laryngeal cancers). The controls were black males of similar age and year of death and were twice the number of cases. Identifying information from death certificates was used to locate the next of kin or close friend of the subjects with esophageal cancer and of the controls for interview. For the purposes of this paper the respondent will be referred to as the next of kin.

Personal interviews of the next of kin were conducted in 1979 by local interviewers under the supervision of a professional survey organization. The interviewers were unaware of the case-control status of the study subjects. The questionnaire used sought information on usual lifetime tobacco consumption (cigarettes, cigars, pipes, chewing tobacco, and snuff); usual lifetime alcohol consumption (beer, wine, and hard liquor) prior to 1974 (i.e., prior to onset of disease); other beverage consumption (carbonated beverages, coffee, and tea); usual dietary patterns during adult life prior to 1974 (frequency of consumption of certain hot spices and sauces, 31 food items, unusual substances eaten, number of meals per day, and methods of cooking); usual adult weight prior to 1974; medical and dental history; lifetime occupational history; highest level of school completed; and residential history (state of birth, childhood state of residence, and length of time living in D.C.).

Quantitative indices of consumption were calculated for several variables. We estimated average daily ethanol intake, assuming 1 fl oz of beer, wine, and hard liquor yields, respectively, 1.1, 2.9, and 9.4 g ethanol (*3*). We then calculated total ethanol consumption by summing the amounts from all three types of alcoholic beverages. The summation was then converted into hard liquor equivalents for ease of interpretation.

ABBREVIATIONS USED: fl oz=fluid ounce(s); RR=relative risk(s).

[1] Received October 27, 1980; accepted April 1, 1981.

[2] Environmental Epidemiology Branch, Division of Cancer Cause and Prevention, National Cancer Institute (NCI), National Institutes of Health, Public Health Service, U.S. Department of Health and Human Services, Bethesda, Md. 20205.

[3] We thank Dr. Jack White, Director of Cancer Research, Howard University Hospital, for his aid in initiating this study; the D.C. Department of Human Resources for supplying death certification information; Ms. Violette Kasica of NCI for data review; Dr. Linda W. Pickle and Dr. B. J. Stone (NCI) for technical assistance; Westat, Inc., for data collection and preparation; and Mrs. Theresa McKinney for manuscript preparation.

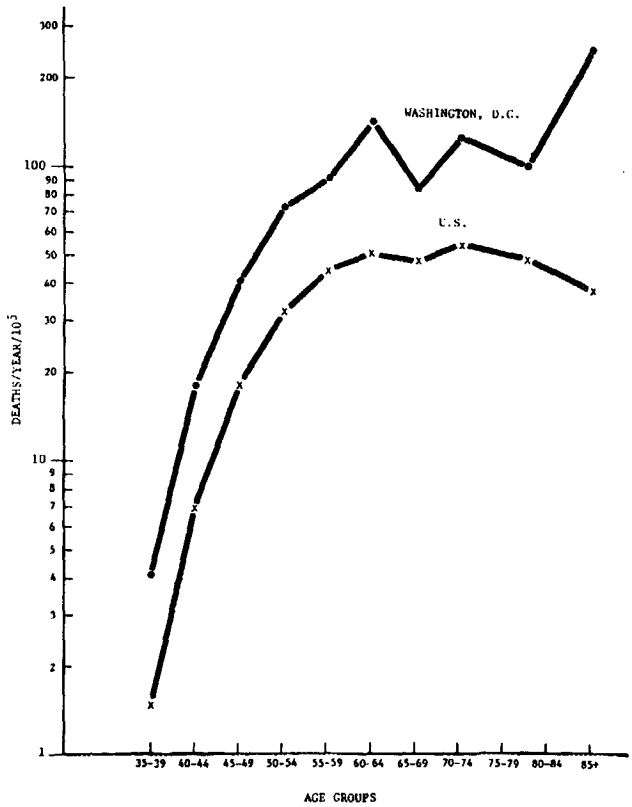
All dietary responses were converted to the number of times the food item was eaten per week. Certain food items were combined to form food groups, such as fresh or frozen meat and fish (beef, chicken, lamb, fish, and shellfish) and precooked or cured meat and fish (bacon, sausage, frankfurters, lunch meat, canned meat, and canned fish). We quantitatively measured the intake of selected micronutrients (e.g., vitamin A and riboflavin) by summing the micronutrient content of each of the food items consumed. Three consumption categories—light, moderate, and heavy—were created for each food item, food group, and micronutrient by the division of the frequency distribution of the variable approximately into thirds.

For the identification of risk factors for esophageal cancer in this population, the interview responses for cases and controls were compared. The measure of strength of association used was the RR, approximated by the odds ratio (*4*). Associations were further examined by calculation of the odds ratios stratified by various factors, particularly ethanol consumption, with summary RR estimated and tested for significance by the Mantel-Haenszel method (*5*) and with confidence intervals calculated as described by Rothman and Boice (*6*). A prospective logistic model was used to adjust for confounding and to test for interaction among risk factors (*7*).

The Mantel extension test (*8*) was used to test risk factors for trend. For alcohol consumption, known to be causally related to esophageal cancer (*9*), attributable risk estimates and associated approximate confidence limits were also calculated (*10, 11*). Student's *t*-tests were used to compare mean weight and height between cases and controls (*12*).

RESULTS

Mortality from esophageal cancer among nonwhite males during 1970-75, 1972 excluded, was higher in D.C. than in other large metropolitan areas of the United States and exceeded that for most urban centers by 50% or more (table 1). The age-adjusted rate of 28.6 deaths/year/10^5 was more than double the U.S. rate of 12.4 for nonwhite males and seven times the national rate of 4.1 for white males. The elevation in mortality was apparent at all ages (text-fig. 1). Esophageal cancer in this period accounted for more deaths (279) among D.C. nonwhite males than all cancers except those of the lung (812 deaths) and prostate gland (320 deaths). Among nonwhite males in the suburban counties surrounding D.C., mortality from esophageal cancer was also high. The age-adjusted rates for Montgomery and Prince Georges Counties in Maryland and Arlington and Fairfax Counties in Virginia were, respectively, 19.6, 23.5, 25.9, and 34.8.



TEXT-FIGURE 1.—Age (yr)-specific esophageal cancer mortality rates in 1970-75 for nonwhite males in Washington, D.C., and the United States.

TABLE 1.—*Age-adjusted esophageal cancer mortality rates, 1970-75,[a] among nonwhite males in the 10 U.S. locations with the largest nonwhite populations*

Location[b]	No. of deaths	Mortality rate, death/yr/10^5
Washington, D.C.	279	28.6
Baltimore City, Md.	153	20.0
Essex County, N.J. (Newark)	78	19.0
New York City, N.Y.	499	17.1
Cook County, Ill. (Chicago)	334	16.4
Philadelphia, Pa.	202	15.7
Cuyahoga County, Ohio (Cleveland)	93	14.7
Wayne County, Mich. (Detroit)	198	13.7
Harris County, Tex. (Houston)	70	11.3
Los Angeles, Calif.	176	10.3

[a] Deaths (and populations) for the yr 1972 were excluded because all deaths in this yr were not recorded by the National Center for Health Statistics.
[b] Major city *in parentheses.*

During 1975-77, 190 deaths among black male residents of D.C. were attributed to primary esophageal cancer. These individuals comprised the eligible case group for the interview study. The controls numbered 380 and represented deaths from the following causes: other cancer (28%), heart and circulatory diseases (42%), cirrhosis (8%), respiratory diseases (4%), accidental or violent deaths (6%), and other causes (12%).

Interviews were completed for 67% of the cases and 71% of the controls. The primary reasons for no interview were the inability to locate the next of kin (19%) and respondent refusal to be interviewed (7%).

Information was considered to be incomplete for 4.5% of the interviews. These were excluded from the analysis, as well as the 2.5% of the study subjects who had lived in Washington, D.C., less than 4 years. The final study population on which the analyses were based, therefore, consisted of 120 cases and 250 controls. The next of kin interviewed for cases and controls, respectively, were wives (45%, 45%), relatives (siblings, children, parents, and other relatives) (48%, 48%), and friends (6%, 7%). Most of the study subjects were longtime residents of the D.C. area (median, 39 yr). The median ages for the cases and controls were 59 and 60 years, respectively.

The major risk factor for esophageal cancer was alcoholic beverage consumption. Ninety-six percent of the subjects with esophageal cancer drank some type of alcoholic beverage compared to 78% of the controls (RR, 6.4; $P<0.001$), yielding an attributable risk of 81% (≈95% confidence interval, 52-91%) from alcohol consumption.

The RR tended to increase with amount of ethanol consumed (table 2). The RR were 4.0 for those who drank less than 6 fl oz in hard liquor equivalents/day, 5.5 for those who drank 6-15 fl oz/day, and 7.6 for those who drank more than 15 fl oz/day, a significant ($P<0.001$) trend. The RR was highest for those who drank hard liquor; however, the RR were also elevated for those who drank wine only, wine and/or beer only, and beer only (table 3). The higher RR asociated with hard liquor use was not solely related to higher ethanol content. As shown in table 4, the RR for hard liquor drinkers of less than 6 fl oz/day was higher than that for wine and/or beer drinkers who drank the ethanol equivalent of 6 or more fl oz of hard liquor/day. The subjects with esophageal cancer drank hard liquor in straight rather than mixed form more often than did the controls (68 vs. 57%). Whiskey (including bourbon) was drunk by a greater percentage of the subjects with esophageal cancer (67%) than by the controls (56%).

The RR for cigarette smoking according to amount smoked are presented in table 5. Eighty-three percent of the subjects with esophageal cancer and 79% of the controls were reported to have smoked cigarettes during their lifetime. Smokers were at increased risk [RR=1.3 (0.7, 2.3); $P=0.65$], with the greatest risk [RR=1.6 (0.8, 3.3)] for those usually smoking two or more packs per day. These risks faded after adjustment for ethanol consumption [overall RR=1.0 (0.5, 1.8)] (table 5). However, the ethanol-associated risk remained high after we controlled for cigarette smoking. For the nonsmokers (20 cases, 53 controls), the RR associated with consumption of alcoholic beverages was 19.9 (2.4, 166.4). The RR for cigarette smoking were also calculated with the use of only the controls whose causes of death were not known to be related to smoking. Thus lung, pancreas, and bladder cancers, heart disease, and chronic lung diseases were excluded. Their deletion raised the overall RR for smoking to 1.9 (1.0, 3.5) (table 5). However, the overall RR was reduced to 1.5 (0.7, 3.0) when adjusted for ethanol consumption, and the trend with increasing amounts smoked was marginally significant ($P=0.06$).

TABLE 2.—*RR of esophageal cancer by daily amount of ethanol consumed in hard liquor equivalents*

Hard liquor equivalents, fl oz/day	No. of cases[a]	No. of controls[a]	RR[b] (95% confidence interval)
None[c]	5	55	1.0 —
1.0–5.9	16	44	4.0 (1.4–12.0)
6.0–14.9	25	50	5.5 (2.0–15.0)
15.0–29.9	25	36	7.6 (2.7–22.0)
30.0–80.6	19	28	7.5 (2.5–22.0)

[a] Excludes 4 cases and 5 controls with unknown drinking status and 26 cases and 32 controls reported to have drunk alcoholic beverages but in unknown amounts.
[b] All risks relative to risks for nondrinkers.
[c] Never drank more than 5 shots or glasses of alcoholic beverages/wk for a period >1 mo.

TABLE 3.—*RR of esophageal cancer by type of alcoholic beverage consumed*

Type of beverage	No. of cases[a]	No. of controls[a]	RR[b] (95% confidence interval)
None[c]	5	55	1.0 —
Any type[d]	111	190	6.4 (2.5–16.4)
Hard liquor only	32	48	7.3 (2.6–20.3)
Hard liquor plus wine or beer	67	106	7.0 (2.7–18.5)
Wine only	2	5	4.4 (0.7–28.8)
Wine and/or beer only[e]	8	29	3.0 (0.9–10.0)
Beer only	3	19	1.7 (0.4–7.8)

[a] Excludes 4 cases and 5 controls with unknown drinking status.
[b] All risks relative to risks for nondrinkers.
[c] Never drank more than 5 shots or glasses of alcoholic beverages/wk for a period of >1 mo.
[d] Includes 4 cases and 7 controls reported to have drunk alcoholic beverage, but information incomplete.
[e] Includes persons who drank wine only, beer only, or both wine and beer.

TABLE 4.—*RR of esophageal cancer according to type and amount of alcoholic beverages consumed*

Hard liquor equivalents, fl oz/day	Alcoholic beverage	No. of cases[a]	No. of controls[a]	RR[b] (95% confidence interval)
0.1–5.9	Wine and/or beer only[c]	4	17	2.6 (0.6–10.8)
	Hard liquor only	6	11	6.0 (1.6–23.2)
	Hard liquor plus wine or beer	6	16	4.1 (1.1–15.2)
≥6.0	Wine and/or beer only[c]	4	12	3.7 (0.9–15.9)
	Hard liquor only	17	28	6.7 (2.2–20.0)
	Hard liquor plus wine or beer	48	74	7.1 (2.6–19.0)

[a] Excludes 30 cases and 37 controls with unknown amount of alcoholic beverages consumed.
[b] All risks relative to risks for nondrinkers.
[c] Includes persons who drank wine only, beer only, or both wine and beer.

TABLE 5.—*RR of esophageal cancer by cigarette smoking categories*[a]

Amount smoked/day	No. of cases	No. of controls	RR[b]	RR adjusted for ethanol consumption
Nonsmoker[c]	20	53 (30)	1.0 (1.0)	1.0 (1.0)
Smoker[d]	99	195 (78)	1.3 (1.9)	1.0 (1.5)
<½ pack	12	25 (13)	1.3 (1.4)	1.1 (1.0)
½–1½ packs	56	117 (51)	1.3 (1.7)	0.8 (1.2)
≥2 packs	22	37 (10)	1.6 (3.3)	0.9 (2.1)

[a] *Numbers in parentheses* refer to numbers of controls with non-smoking-related causes of death and corresponding RR.
[b] All risks relative to risks for nonsmokers.
[c] Never smoked 100 or more cigarettes during their lifetime.
[d] Includes 9 cases and 16 controls known to have smoked but in unknown amounts. Excludes 1 case and 2 controls with unknown smoking status.

A comparison of other forms of tobacco revealed no increases in risk. Only a small percentage of the subjects with esophageal cancer had smoked cigars (14%), smoked pipes (13%), chewed tobacco (3.3%), or used snuff (1.7%). For each of these forms of tobacco, except the last, the percentage of ever users was slightly higher among the controls. However, 13 of the 20 subjects with esophageal cancer who did not smoke cigarettes smoked pipers or cigars compared to 22 of the 53 nonsmoking controls. The RR for pipe or cigar smoking among noncigarette smokers was 2.6 (0.9, 7.6).

Differences in food consumption were observed between cases and controls. The subjects with esophageal cancer ate more bacon, sausage, frankfurters, lunch meat, canned meat, canned fish, liver, and potatoes but less beef, chicken, lamb, fish, eggs, milk, vegetables, and fruit. As can be seen in table 6, a greater proportion of the subjects with esophageal cancer than the controls ate fewer than three meals a day [RR = 1.8 (1.1, 3.0)] but slightly more precooked or cured meat and fish. The RR associated with low compared to high consumption of 1) fresh or frozen meat and fish, 2) dairy products and eggs, and 3) fruits and vegetables were about twofold and were not substantially altered when adjusted for ethanol consumption or level of education. While the RR for consumption of vitamin A, vitamin C, riboflavin, and thiamine increased with decreased intake, the trends for the 3 food groups were more impressive. No clear association with nitrite-containing foods was observed. A trend of increasing RR was seen with a decrease in relative weight, although very few of the subjects with esophageal cancer were especially light in weight. The average adult weight for the cases (162.8 lb) was significantly less than that of the controls (171.0 lb) ($P = 0.009$), but their average heights were similar, 68.4 and 68.1 inches, respectively.

Analyses with the use of linear logistic models that simultaneously considered the factors of age, alcohol, smoking, and diet tended to show independent effects associated with the nutritional indices. For example, we found the RR for consumption of moderate and low levels of fresh or frozen meat and fish to be 1.6 (0.85, 3.0) and 2.3 (1.1, 4.4), respectively, after adjusting for these other factors. This same analysis yielded RR for the five ethanol categories (table 2) of 1.0, 4.2 (1.4, 13.0), 6.5 (2.2, 19.5), 8.1 (2.7, 24.2), and 9.2 (2.9, 29.1).

The RR for consumption of hot spices and sauces, such as chili peppers, red peppers, and hot sauce, revealed no significant trends for the four levels of consumption—never, rarely, occasionally, and often. No significant differences were observed between cases and controls in the consumption of carbonated beverages, coffee (any and "burning hot"), and tea (any, burning hot, and herbal). No significant case-control differences were observed for the following dental

TABLE 6.—*RR of esophageal cancer by selected nutritional indices*

Nutrition index	RR: Consumption level			RR adjusted for ethanol, consumption level			RR adjusted for education,[a] consumption level		
	High	Moderate	Low	High	Moderate	Low	High	Moderate	Low
Food groups									
Fresh or frozen meat and fish	1.0	1.5	2.1[b]	1.0	1.6	2.2[b]	1.0	1.6	2.1[b]
Dairy products and eggs	1.0	1.6	2.0[b]	1.0	1.7	1.9[b]	1.0	2.0	2.1[b]
Fruits and vegetables	1.0	2.1	2.4[c]	1.0	1.7	2.0[b]	1.0	2.0	2.8[c]
Precooked or cured meat and fish	1.0	0.9	0.8	1.0	0.9	0.9	1.0	0.8	0.6
Nitrite-containing foods	1.0	1.1	0.8	1.0	1.1	1.0	1.0	1.0	0.7
Micronutrients									
Vitamin A	1.0	1.4	1.5	1.0	1.5	1.5	1.0	1.4	1.4
Vitamin C	1.0	1.3	2.1[c]	1.0	1.2	1.8[b]	1.0	1.2	2.1[c]
Riboflavin	1.0	1.1	1.6	1.0	1.0	1.7[b]	1.0	1.2	1.7
Thiamine	1.0	1.2	1.1	1.0	1.2	1.2	1.0	1.2	1.1
Other									
Relative wt	1.0	1.6	2.4[c]	1.0	1.5	2.1[c]	1.0	1.7	2.6[c]
Meals per day	1.0	—	1.8	1.0	—	1.6	1.0	—	1.9

[a] Excludes persons with unknown educational status.
[b] Significant ($P<0.05$) trend.
[c] Significant ($P<0.01$) trend.

health indices: the condition of teeth and gums, the number of teeth lost due to decay, the wearing of false teeth, and the number of times permanent teeth were brushed per week. Also, there were no clear case-control differences for ever having had anemia, arthritis, or thyroid disease. Review of the occupational histories revealed no significant differences when jobs were grouped by major industry (e.g., construction, transportation, and military and other government services) and by level of skill (e.g., unskilled, skilled, or professional).

The esophageal cancer subjects tended to have less formal education than did the controls. There was a trend of increasing risk with decreasing level of education (RR = 1.0, 1.3, and 1.5, respectively, for ≥12, 8-11, and <8 yr of school completed). These risks were not affected by the adjustment for ethanol consumption.

Approximately half of our study population was born in the D.C. area, including parts of Maryland and Virginia. A higher percentage of the subjects with esophageal cancer than the controls was born in either Georgia [RR = 1.3 (0.8, 2.8)] or North Carolina [RR = 1.5 (0.5, 3.3)], but there was no excess risk associated with either being born or spending one's childhood in South Carolina, where a cluster of high rates for esophageal cancer has been identified (*13*).

DISCUSSION

Esophageal cancer varies more worldwide than any other neoplasm, with annual rates exceeding 100 per 100,000 population for both males and females in parts of northern China, Iran, and the Soviet Union (*14*). Reasons for the exceptional risk in these areas are obscure. In western countries, the major risk factors are alcohol consumption and cigarette smoking, which probably account for the higher rates in cities and in males (*15, 16*). In the United States, the rates are much higher in blacks than in whites, particularly in the urban areas (*16*). The present study was conducted among black men in Washington, D.C., where the death rate for esophageal cancer ($28.6/10^5$) is especially high, exceeding the national level for nonwhite males by over twofold and for white males by sevenfold.

The major risk factor was found to be ethanol consumption (RR = 6.4), which was estimated to be causally associated with about 80% of the neoplasms among the esophageal cancer subjects.[4] Nearly all the esophageal cancer subjects drank alcoholic beverages, usually in very high quantities.

The controls drank more than expected when compared to males of the populations surveyed in other studies. Forty-six percent consumed six or more alcoholic drinks per day, whereas only 4.4% of the 5,000 black males enrolled in the Kaiser-Permanente health plan in the Oakland-San Francisco area drank this amount (*18*). In addition, a national survey conducted in 1972-74 showed that less than 15% of males 40 years of age or over drank 1 or more oz of ethanol/day (*19*). These data suggest that blacks in D.C. drink more than blacks in other areas of the United States. Although our study differed from other studies in the phrasing of the questions asked, in second-party versus direct reporting, and in obtaining information about deceased rather than about living individuals, such study differences seem unlikely to account for the substantially higher consumption reported in D.C. Other evidence exists of high alcoholic beverage consumption in D.C. The per-capita "apparent consumption" of alcoholic beverages on the basis of tax revenues for D.C. surpasses the national level by nearly fourfold for hard liquor and about threefold for wine (*19*), although part of the excess is related to purchases by nonresidents. In addition, the computation of age-adjusted mortality rates from cirrhosis of the liver for the years 1965-71 revealed that nonwhite males in D.C. had rates about 2.5 times higher than those of U.S. nonwhite males.

Although an increased risk of esophageal cancer in this study was associated with all forms of alcoholic beverages, the excess was greatest for hard liquor, particularly whiskey or bourbon. A gradient in risk according to ethanol concentration was evident with straight liquor having the highest relative risk, mixed liquor and wine having an intermediate risk, and beer having the lowest risk. This finding, together with the gradient in risk observed with increasing amounts of ethanol consumed, is consistent with a causal effect of alcohol consumption.

Cigarette smoking is regarded as a major risk factor for esophageal cancer, and some studies have shown a synergism between tobacco and ethanol consumption (*14*). In contrast, the overall data from our case-control study revealed no significant risk associated with cigarette smoking and also no consistent enhancement of risk following exposure to both alcoholic beverages and cigarette smoking. The discrepancy in part relates to different comparison groups. Controls in our study included persons who died from lung cancer and heart disease, conditions known to be induced by cigarette smoking, whereas some other studies (*15*) excluded as controls persons who died from smoking-related conditions. When we restricted our comparisons by using only those controls who died of illnesses not known to be linked to smoking, a 90% increased risk of esophageal cancer and a dose-response relationship were observed for smokers. However, this smoking-associated risk was considerably reduced when we controlled for ethanol intake. Possibly, the consumption of such large amounts of alcoholic beverages by our study population overwhelmed the risk that would have been due to cigarette smoking in a population consuming lesser amounts.

[4] It is likely that the consumption of alcoholic beverages among our mortality series of controls is greater than that of the living Washington, D.C., black male population inasmuch as drinking contributes to many causes of death. If this is the case, then the relative and attributable risks of esophageal cancer due to ethanol consumption would actually be higher in a population of living subjects than calculated for our study group.

Whereas esophageal cancer has been related to pipe smoking, cigar smoking, tobacco chewing, and other smokeless tobacco products in various populations (*14*), the percentage of D.C. residents using a tobacco product other than cigarettes was small. Only among noncigarette smokers was an association found with pipe and cigar smoking.

Review of the dietary information revealed an increased risk of esophageal cancer associated with poor nutritional status. The esophageal cancer subjects were approximately the same height as controls but their usual adult weight before onset of cancer was less, although very few were seriously underweight. They were reported to eat fewer meals per day and less dairy products and eggs, fruits and vegetables, and fresh or frozen meat and fish. Although food intake can be limited by alcoholic beverage consumption, twofold differences between high and low consumption of the food variables were seen after adjustment for ethanol and social staus. Specific indices of vitamin A, vitamin C, riboflavin, and thiamine showed patterns similar to the general nutritional measures, although of a lower order of magnitude. These findings, consistent with those of other studies (*14*), suggest that poor nutrition, possibly involving complex dietary deficiencies, is involved in the development of esophageal cancer.

These results may be influenced by a case recall bias. Despite an attempt to assess dietary patterns predating the disease (i.e., prior to 1974), it is possible that the next of kin recalled a decrease in food consumption caused by the esophageal cancer instead of by the study subjects' usual lifetime dietary patterns. This possibility seems unlikely, however, since a history of low food intake among the esophageal cancer subjects did not apply to all food items (e.g., the esophageal cancer subjects did eat more of the precooked and cured varieties of meat and fish), and these esophageal cancer subjects were not found to be underweight when compared to black males 55-64 years of age who participated in the 1971-74 U.S. Health and Nutrition Examination Survey (Abraham S: Personal communication). Furthermore, although knowledge of the diets of cases and controls by next of kin may have been incomplete, when the RR for each of the major food groups were calculated separately by respondent type, the RR determined from the wife's responses (considered to be most accurate) showed good agreement with the RR determined from the responses of other relatives.

Information was obtained on several potential risk factors (including consumption of coffee, tea, and burning hot liquids; history of certain medical conditions; and occupation), but no significant case-control differences were observed. While the present study showed no association between poor oral hygiene and increased risk of esophageal cancer, next-of-kin responses may not have been adequate to assess the dental health of the study subjects. One hypothesis under test concerned migration from other areas, especially coastal South Carolina where mortality from esophageal cancer has been extremely high among blacks at least since the early 1950's (*17*). A higher proportion of esophageal cancer subjects than controls was born in the Deep South, but there was no excess of migrants from South Carolina.

In this case-control study, with information obtained from next of kin of deceased patients, we had an overall response rate of 70%. Nonresponse was primarily related to difficulty in locating the next of kin of the decedents. Although information is limited on the characteristics of the nonrespondents, we suspect that they may have represented study subjects who were poor and without close friends or family ties. This type of response bias might affect the comparisons made but would not materially reduce the RR presented. We made the decision to interview relatives because esophageal cancer is a highly fatal disease [the median survival for blacks nationwide is 4 mo (*20*)] and because establishing a rapid reporting system for interviewing a high percentage of newly diagnosed patients proved not to be feasible. Lack of knowledge or difficulty in recall by the next of kin may have influenced the responses but probably not differentially between cases and controls because the questions were asked in a similar manner by interviewers "blind" to the disease status of the study subjects. Comparison of self versus next-of-kin interviews in other studies has revealed generally good concordance for broadly defined variables, including smoking (*21-23*), alcohol intake and dietary history (*21*), and usual occupation (*22*).

Despite the limitations of a case-control study involving next-of-kin respondents in a difficult-to-locate population, the data gathered appeared adequate to identify alcoholic beverage consumption as the major factor responsible for the elevated risk of esophageal cancer among black males in D.C. and also to contribute some intriguing hypotheses regarding the role of poor nutrition in the origins of this cancer.

REFERENCES

(*1*) MASON TJ, MCKAY FW. U.S. cancer mortality by county 1950-69. Washington, D.C.: U.S. Govt Print Off, 1973 [DHEW publication No. (NIH)74-615].

(*2*) National Center for Health Statistics. International Classification of Diseases. Eighth revision. Washington, D.C.: U.S. Govt Print Off, 1967 (PHS publication No. 1693).

(*3*) ADAMS CF. Nutritive value of American foods in common units. Washington, D.C.: U.S. Govt Print Off, 1975 (USDA agriculture handbook No. 456).

(*4*) FLEISS JL. Statistical methods for rates and proportions. New York: Wiley, 1973.

(*5*) MANTEL N, HAENSZEL W. Statistical aspects of the analysis of data from retrospective studies of disease. J Natl Cancer Inst 1959; 22:719-748.

(*6*) ROTHMAN KJ, BOICE JD. Epidemiologic analysis with a programmable calculator. Washington, D.C.: U.S. Govt Print Off, 1979:5-6. [U.S. DHEW publication No. (NIH)79-1649].

(*7*) PRENTICE RG, PYKE R. Logistic incidence disease models and case-control studies. Biometrika 1969; 66:408-412.

(*8*) MANTEL N. Chi-square tests with one degree of freedom; extensions of the Mantel-Haenszel procedure. J Am Stat Assoc 1963; 58:690-700.

(*9*) ROTHM
risk
New
(*10*) MACMA
Bost
(*11*) WALTE
risk
(*12*) SNEDEC
Iowa
(*13*) BATES
the e
453-
(*14*) DAY NE
D, Fr
Phila
(*15*) WYNDE
of th
(*16*) SCHOEN
tality
1930-
(*17*) FRAUME

(9) ROTHMAN KJ. Alcohol. In: Fraumeni JF Jr, ed. Persons at high risk of cancer: An approach to cancer etiology and control. New York: Academic Press, 1975:139-148.
(10) MACMAHON B, PUGH TF. Epidemiology principles and methods. Boston: Little, Brown & Co., 1970:273-275.
(11) WALTER SD. The estimates and interpretation of attributable risk in health research. Biometrics 1976; 32:829-849.
(12) SNEDECOR GW, COCHRAN WG. Statistical methods. Ames, Iowa: Iowa State Univ Press, 1967:59-60.
(13) BATES DC, CASTON JC, O'BRIEN P, SANDIFER SH. Carcinoma of the esophagus in South Carolina. J SC Med Assoc 1971; 67: 453-456.
(14) DAY NE, MUNOZ N. Cancer of the esophagus. In: Schottenfeld D, Fraumeni JF Jr, eds. Cancer epidemiology and prevention. Philadelphia: Saunders. In press.
(15) WYNDER EL, BROSS IJ. A study of etiological factors in cancer of the esophagus. Cancer 1961; 14:389-413.
(16) SCHOENBERG BS, BAILAR JC III, FRAUMENI JF JR. Certain mortality patterns of esophageal cancer in the United States, 1930-67. J Natl Cancer Inst 1971; 46:63-73.
(17) FRAUMENI JF JR, BLOT WJ. Geographic variation in esophageal cancer mortality in the United States. J Chronic Dis 1977; 30: 759-767.
(18) KLATSKY AL, FRIEDMAN GD, SIEGELAUB AB, GERALD MJ. Alcohol consumption among white, black or oriental men and women; Kaiser Permanente multiphasic health examination data. Am J Epidemiol 1977; 105:311-322.
(19) KELLER M, ed. Second special report to the U.S. Congress on alcohol and health. Washington, D.C.: U.S. Govt Print Off, 1975 [DHEW publication No. (ADM)75-212].
(20) AXTELL LM, ASIRE AJ, MYERS MH, eds. Cancer patient survival. Washington, D.C.: U.S. Govt Print Off, 1976 [Report No. 5. DHEW publication No. (NIH)77-992].
(21) KOLONEL LM, HIROHATA T, NOMURA AM. Adequacy of survey data collected from substitute respondents. Am J Epidemiol 1977; 106:476-483.
(22) ROGOT E, REID DD. The validity of data from next-of-kin in studies of mortality among migrants. Int J Epidemiol 1975; 4: 51-54.
(23) BLOT WJ, HARRINGTON JM, TOLEDO A, HOOVER R, HEATH CW JR, FRAUMENI JF JR. Lung cancer after employment in shipyards during World War II. N Engl J Med 1978; 299:620-624.